Bleeding disorders p

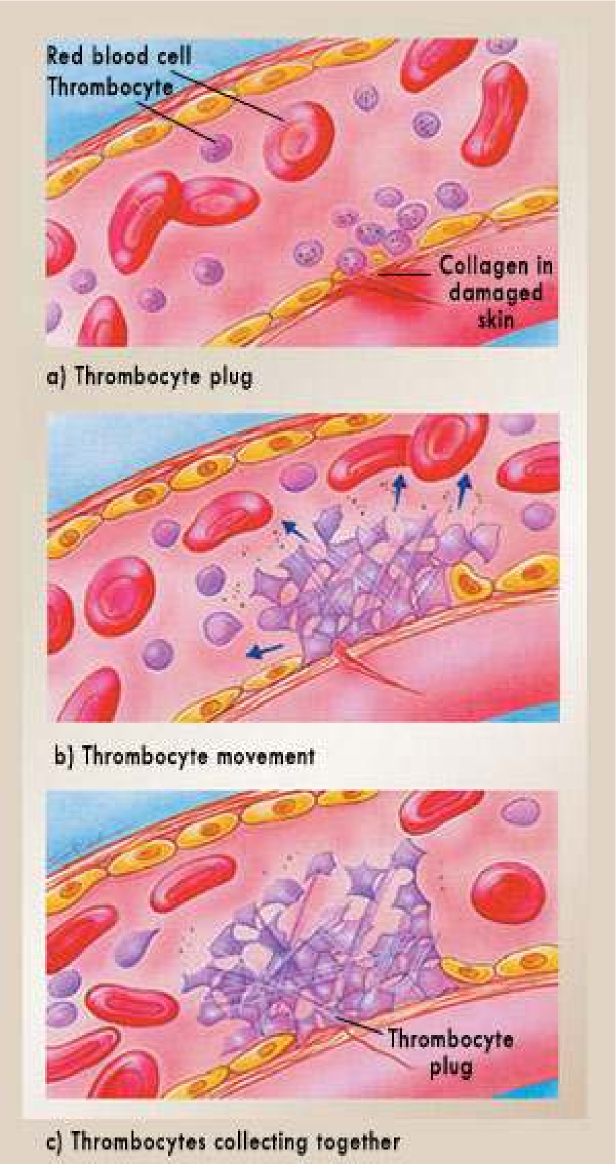
BLEEDING AND CLOTTING DISORDERS

PATHOPHYSIOLOGY

-The coagulation cascade is underway within 10-20 sec of injury, an initial hemostatic plug is formed in 1-3 minutes, and fibrin has been generated and added to stabilize the clot by 5-10 minutes

Phases of coagulation

1)Vascular phase
2) Platelet phase
3) Coagulation phase
4) Fibrinoytic phase



a) Thrombocyte plug

b) Thrombocyte movement

c) Thrombocytes collecting together

Vascular phase

-After tissue injury, there is an immediate reflex vasoconstriction in small vessels

-Reactant such as serotonin, histamine and prostaglandin are vasoactive and produce vasoconstriction

Platelet phase

1) platelet are exposed to damaged vascular surfaces
2) they undergo aggregation and form the primary vascular plug that reduce blood loss from small blood vessels
3) the platelet plugs adhere to exposed basement membrane

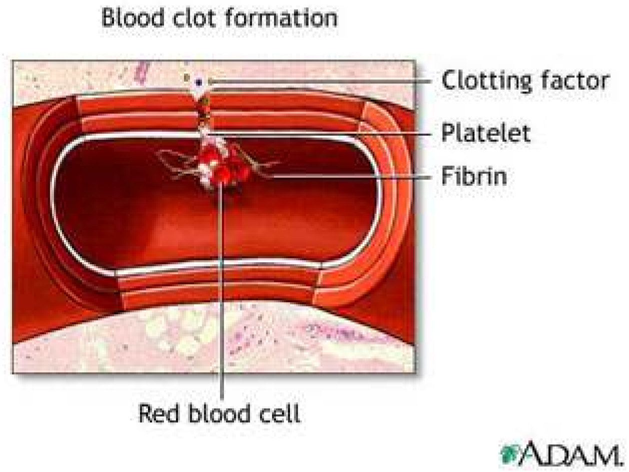
Goagulation phase

- The process of coagulating essentially involves three pathways (intrinsic and extrinsic) that converge by activating a third (common pathway)

Clinical manifestations

-mild diseases =may present with no clinical signs

-when skin and mucosa are involved, petechia, ecchymosis,hematomas or jaundice may be present



Clinical laboratory test

- ***A) Bleeding time***
- ***B)coagulation time***
- C- platelet count

Classification of bleeding disorders

I- vessel wall disorders

Causes:

1) damage to capillary endothilium
2) abnormalities in the vascular subendothelial matrix
3) abnormal vessel formation

II- platelet disorders

- Congenital platelet’s disorders
- Acquired platelet’s disorders

III- coagulation disorders

1) congenital coagulopathies:
    - hemophilia A
    - hemophilia B
    - Von Willibrand’s disease
2) anticoagulant related coagulopathies
    - heparin
    - coumarin

3) disease related coagulopathies;

  - liver disease
  - vitamin K deficiency

4) fibrinolytic disorders

hemophilia

- Hemophilia are rare congenital diseases characterized by severe bleeding tendency
- Types:
- Hemophilia A :classic type transmitted as sex linked recessive disaese affecting male,it is characterized by deficiency of factor VIII(AHG)

Clinical manifestation

- When factor VIII> 1% of normal = severe hemorrhage
- When factor VIII 1- 5% of normal= moderate clinical bleeding is found
- When factor VII 6- 50% of normal = mild symptoms as prolonged bleeding following tooth extraction ,surgical procedure, or severe trauma

2) anticoagulant related coagulopathies

anticoagulants

Coumarin

-Oral:for long term use

-Vit k antagonist:inhibit hepatic synthesis of vit k dependent coagulation factors(2,7,9,10)

Begins to work within 6 hours ,effective by 12hours

-Antagonized by vit k

-Elevates PT

Heparin

-Parental for short term use

-Inhibits thrombin-fibrinogen reaction in large doses,

-it prevent platelets aggregation

-Act immediately for 4-6 hours

-Antagonized by protamine sulphate

-Elevate PT,PTT

3) disease related coagulpathies

Liver disease

- It synthesis most clotting factors(except factor VIII)
- Bile is necessary for the absorption of fat soluble vitamins(A,D,E,K)
- Thus:impaired hepatic cell function→ impaired formation of clotting factor
- Obstructive jaundice →impaired vit K absorption →decreased factor 2,7,9,10 formation by hepatic cells

4- fibrinolytic disorders

Dental management

- Gingival bleeding or oozing:
- Use fibrin foam,gel foam or absorbable cellulose with thrombin,1.5%H2O2 mouth rinse
- Elective surgery:should be referred
- For emergency dental ttt: hospitalized the patient
- Extraction socket are packed with gel foam